REMARKS

I. Substance of the Interview Statement

Applicants thank the Examiner and the Examiner's Supervisor for the interview on January 12, 2011. During the interview, the present claims, the outstanding rejections, and the differences between the present claims and the cited references were discussed.

II. Claim Amendments

By the foregoing amendments to the claims, claims 20 and 36 have been amended to recite that the histone deacetylase inhibitor is a compound of formula (I). Furthermore, the claims have been amended to recite that the method increases transgene expression; to encompass derivatives of formula (I); and to recite that the subject is human. New dependent claims 40 and 41 recite that the histone deacetylase inhibitor is FK228. New dependent claims 42 and 43 recite that the derivative is obtained through acetylation of the compound or through reduction of the S-S bond. These amendments to the claims are supported throughout the application as filed. In particular, methods for increasing transgene expression are supported at least at pages 11 and 15 of the specification as filed; human subjects are supported at least at page 11, lines 10-14; FK228 (a stereoisomer represented by formula (II)) is described at least at page 9; and derivatives of formula (I) are described at least at page 8, lines 9-12.

Additional amendments to the claims have been made for clarity and to bring the claims into better conformance with U.S. patent practice; these editorial amendments are not intended to change the scope of the claims or any elements recited therein.

Finally, claims 21, 25, 28, 30, and 34 have been canceled.

The amendments to the claims, including cancellation of claims, have been made without prejudice or disclaimer to any subject matter recited or canceled herein. Applicants reserve the right to file one or more continuation and/or divisional applications directed to any canceled subject matter. No new matter has been added, and entry of the foregoing amendments to the claims is respectfully requested.

III. Response to Claim Rejections Under 35 U.S.C. § 102

Claims 20, 24, and 36 have been rejected under 35 U.S.C. § 102(b), as allegedly being anticipated by Samulski et al., U.S. Patent No. 6,410,300. This rejection is respectfully traversed.

Not to acquiesce to the rejection, but to advance prosecution, as noted above the claims have been amended to recite that the histone deacetylase inhibitor is a compound of formula (1). Samulski et al. do not teach or even suggest a compound of formula (I). For at least this reason, the cited reference does not teach each and every element of the present claims. Accordingly, Applicants respectfully request reconsideration and withdrawal of this rejection.

IV. Response to Claim Rejections Under 35 U.S.C. § 103

Claims 20, 21, 24-26, 33, 34, 36, and 38 have been rejected under 35 U.S.C. § 103(a) as allegedly being unpatentable over Samulski et al. in view of Kitazono et al., Nakajima et al., and Alisky et al. This rejection is respectfully traversed.

Samulski et al. teaches administering sodium butyrate to upregulate the expression of cell surface AAV receptors, thus facilitating infection of AAV into a host cell (col. 14, lines 28-37). However, Samulski et al. does not teach or suggest administering a compound of formula (I).

With regard to Kitazano et al., this reference is directed to adenoviral vectors rather than to AAV vectors. Kitazono et al. teach that treatment of cells with the histone deacetylase inhibitor FR901228 (depsipeptide) increases CAR and α_5 integrin levels. A person of ordinary skill in the art would have recognized that while CAR and α_5 integrin are cell surface receptors for adenovirus, these receptors do not play a role in AAV binding and entry. Thus, the skilled person would not have been motivated to combine Samulski et al. and Kitazono et al. with any reasonable expectation of success.

Furthermore, Applicants respectfully submit that the method recited in the present claims provides unexpected results as compared to the prior art. In particular, Kitazano et al. demonstrate that treatment with sodium butyrate, trichostatin A (TSA), and other histone deacetylase inhibitors "cause a similar increase" in adenoviral receptor RNA levels as compared to FR901228. In contrast, Figure 3A of Okada et al. (Molecular Therapy 13:738-746, 2006 (of record)) shows that treatment with a compound of Formula (I) (i.e. FR901228) significantly

improves AAV transgene expression in a human glioma cell line as compared to other unrelated histone deacetylase inhibitors, such as TSA and FR901464.

Finally, Applicants submit that Nakajima et al., and Alisky et al. do not remedy the serious deficiencies of Samulski et al. in view of Kitazono et al.

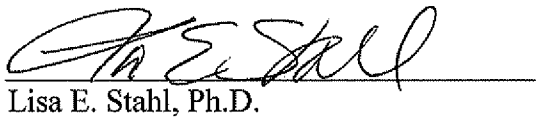
In view of the above, Applicants respectfully request reconsideration and withdrawal of this rejection.

CONCLUSION

In view of the foregoing, further and favorable action in the form of a Notice of Allowance is believed to be next in order. Such action is earnestly solicited.

In the event that there are any questions related to this response, or the application in general, it would be appreciated if the Examiner would telephone the undersigned attorney at the below-listed telephone number concerning such questions so that prosecution of this application may be expedited.

Respectfully submitted,

By: 
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